## **Historic, Archive Document**

Do not assume content reflects current scientific knowledge, policies, or practices.



PESTS NOT KNOWN TO OCCUR IN THE UNITED STATES OR OF LIMITED DISTRIBUTION, NO. 55: BROWN STRIPE DOWNY MILDEW

Prepared by A. Green, Biological Assessment Support Staff, PPQ, APHIS, USDA, Federal Building Room 634, Hyattsville, MD 20782

Disease

BROWN STRIPE DOWNY MILDEW

Pathogen

Sclerophthora rayssiae var. zeae Payak and Renfro

Class:

Oomycetes: Peronosporales: Peronosporaceae

Economic Importance

Order: Family

The downy mildews are primarily diseases of Old World origin that have proved damaging to corn, a New World crop. Brown stripe downy mildew was first identified as a distinct disease by Payak and Renfro (1967). Sclerophthora rayssiae var. zeae caused 20-70 percent infection in many corn-growing areas of India with an incidence of 80-100 percent in certain high hilly tracts (Singh 1971b). Annual losses from this disease in India have been estimated in the millions of dollars (Frederiksen and Renfro 1977).

Hosts

Zea mays (corn) is the primary host of brown stripe downy mildew. The weed <u>Digitaria sanguinalis</u> (large crabgrass) is susceptible (Frederiksen and Renfro 1977).

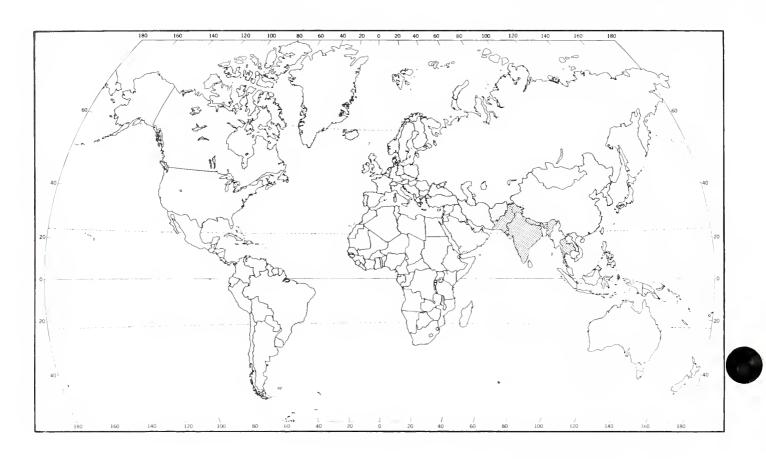
General Distribution The disease is widespread in India (including Sikkim). In recent years it has also been reported in Nepal, Pakistan, and Thailand (Frederiksen and Renfro 1977).

Characters

ASEXUAL STAGE - Sporangia borne in groups of 2-6 on determinate sporangiophores which emerge by way of stomata. Sporangia hyaline, ovate or cylindrical, and 29-66 X 18-26 µm. Each germinating sporangium can produce 10-16 zoospores (motile, flagellated spores). Zoospores spherical, hyaline, 8-11 µm in diameter (Payak and Renfro 1967, Payak, Renfro, and Lal 1970, Singh 1971b).

SEXUAL STAGE - Thin-walled, straw-colored oogonia (female gametangia) scattered through the mesophyll of the host leaves are  $29-44~\mu m$  in diameter. Antheridia (male gametangia) tightly pressed to oogonia.

Single thick-walled oospore forms within oogonia, spherical, or subspherical, hyaline, 29-37  $\mu$ m in diameter, and with prominent oil globule.



Sclerophthora rayssiae var. zeae distribution map prepared by Non-Regional Administrative Operations Office and Biological Assessment Support Staff, PPQ, APHIS, USDA

Characteristic Damage Sclerophthora rayssiae var. zeae attacks the leaves, leaf sheaths, and seeds of corn. Mycelia of the pathogen have not been observed in the stalks or roots of the host. Symptoms first appear on lower leaves, then progress up the plant. The first foliar symptoms are small chlorotic flecks (Fig. 1). These grow into narrow (3-7 mm), parallel yellow lesions with clear margins delimited by leaf veins. The lesions expand longitudinally and turn purple to brown giving the leaf a striped or blotched appearance (Fig. 2). Older leaves can turn completely brown (Payak, Renfro, and Lal 1970). Infection in the seedling stage can suppress cob and seed development and cause premature death of the plant (Singh 1971b).

(Figs. 1-2)





2

Sclerophthora rayssiae var. zeae on corn leaves: 1. Early symptoms (Courtesy M. M. Payak). 2. Parallel lesions (Courtesy B. L. Renfro).

Mycelia of the pathogen can infect the embryo of the seed and systemically attack the germinating seedling. Chlorotic leaf lesions develop within 6-9 days after germination of infected seeds (Singh, Joshi, and Chaube 1968).

The downy growth which characterizes downy mildew diseases is caused by the numerous sporangia and sporangiophores that grow through the stomata of the host leaf. The sporangia of S. rayssiae var. zeae can be observed on the leaf lesions on both surfaces of the leaf.

Brown stripe downy mildew could be confused with two potentially destructive downy mildew diseases of corn already established in the United States. These diseases are caused by the pathogens Sclerophthora macrospora (Sacc.) Thirum. (crazy top) and Peronosclerospora sorghi (Weston and Uppal) C. G. Shaw (sorghum downy mildew).

In crazy top, growth in the tassel proliferates into a mass or tangle of leafy structures (phyllody), creating the "crazy top" appearance. Also, numerous shoots or tillers sprout from the base of the original shoot. Phyllody and excessive tillering are not symptomatic of brown stripe downy mildew.

Sorghum downy mildew causes infected plants to appear chlorotic and frequently stunted. White stripes may appear on leaves. Also, the leaves are narrower and more erect than on healthy plants. Phyllodied tassels may be present. None of these symptoms are observed in brown stripe downy mildew (Shurtleff 1980).

Sclerophthora rayssiae var. rayssiae Kenneth, Koltin, and Wahl causes a downy mildew disease in barley (Hordeum sp.). This pathogen closely resembles S. rayssiae var. zeae, but they differ in host range (Payak, Lal, and Renfro 1970).

Detection Notes

- 1. The entry of corn plant parts and seed from foreign localities where brown stripe downy mildew is known to occur is regulated by Title 7, Part 319.24 and Part 319.41 of the Code of Federal Regulations. Corn seed imported for planting purposes is prohibited entry from these localities. Corn seed imported from these localities for purposes other than planting may enter only with a USDA permit and mandatory sterilization by a USDA approved treatment.
- 2. For field detection, look for parallel leaf lesions, purple to brown, with clearly defined margins, the most characteristic symptoms of brown stripe downy mildew on corn. Eventually, the entire leaf may take on a burnt-brown appearance.
- 3. Check for downy growth on both sides of the infected leaf, most commonly observed on cool, damp mornings. This downy growth will usually disappear by the late afternoon.
- 4. Notice that infected leaves remain intact. Leaf shedding has not been observed in this disease (Singh 1971b).

5. Positive identification of S. rayssiae var. zeae is based on the symptoms and the physical characters of the oogonia, oospores, and sporangia (Payak and Renfro 1967). Submit for identification, dried, pressed leaf samples with well-developed lesions. All disease material should be packed in double containers (one container inside another) with screw lids.

Biology and Etiology The overwintering stage of <u>S. rayssiae var. zeae</u>, the oospore, initiates the disease cycle. Oospores remain viable in dry leaf matter for at least 4 years in the laboratory (Singh 1971a). In the field, oospores are found in plant debris and in the soil.

The oospore produces a sporangiophore bearing a sporangium. The sporangia germinate by either producing zoospores or by directly forming a germ tube. The zoospores are the propagules which usually accomplish the primary infection. Occasionally, a sporangial germ tube infects the corn leaf (Payak, Renfro, and Lal 1970).

The germ tubes of the zoospores penetrate the host leaf through the stomata (Singh 1971b). Disease symptoms appear on the corn leaf 3 days after inoculation with zoospores under favorable environmental conditions (Singh, Renfro, and Payak 1970).

Following infection, coenocytic mycelia (without septations) grow intercellularly in the mesophyll tissue of the host leaf. No haustoria have been observed.

Once the disease is established, sporangia generate the inoculum for secondary infection. They are borne on sporangio-phores which extend through the stomata to the leaf surface. Under optimal conditions of moisture and temperature sporangia germinate and produce large numbers of potentially infectious zoospores. Sporangia and zoospores are disseminated by wind, rain, water, and physical contact between plants (Singh and Renfro 1971).

In an experiment, infected corn plants placed in a moist chamber following the removal of all sporangia produced a 'second crop' of over 200,000 sporangia per plant in 3-7 hours under optimal temperatures (22-27° C). This indicates the tremendous inoculum production potential of this pathogen (Singh, Renfro, and Payak 1970).

Leaf lesions are a symptomatic result of the growth and sporulation of the fungus. As the lesions begin to age, oogonia and antheridia appear scattered through the mesophyll. Oospores will eventually form in the oogonia, producing the potential disease source for the next crop season (Singh 1971b).

The disease can also be internally seedborne, resulting in the systemic infection of the germinating seedling (Singh, Joshi, and Chaube 1967). The importance of seedborne infection in the epidemiology of this disease, however, has not been demonstrated.

Moisture is probably the most important environmental factor influencing disease development. Twelve hours of continued moisture (75-85 percent humidity in a moist chamber) were necessary for successful infection following inoculation with zoospores. Increasing the wet period up to 72 hours increased the likelihood of infection (Singh, Renfro, and Payak 1970). In India, areas with the highest rainfall, over 100 cm per year, had the highest incidence of brown stripe downy mildew (Payak, Renfro, and Lal 1970).

Temperature also affects epidemiology. Zoospores germinated at  $15-30^{\circ}$  C with an optimal range of  $22-25^{\circ}$  C. Similarly, sporangial germination occurred at  $18-30^{\circ}$  C with an optimal range of  $20-22^{\circ}$  C (Singh, Renfro, and Payak 1970).

The age of the plant at the time of inoculation directly affects disease development. Young plants, 10-30 days old, showed a much higher rate of systemic infection following inoculation with zoospores than plants over 40 days old (Singh, Renfro, and Payak 1970).

Digitaria sanguinalis is a weed which serves as a collateral host of the disease. In one study, naturally infected weeds were growing along the edge of a field plot. Disease incidence was directly and significantly affected by the proximity of the corn rows to the weed host (Bains, Jhooty, and others 1978).

Control

Use of resistant cultivars has been the most effective means of controlling the downy mildew diseases of corn. In India, resistance to brown stripe downy mildew was tested on 2,113 different strains of corn. A total of 58 was highly resistant and 667 were resistant. These results indicate that breeding material is available for the development of resistant cultivars (Singh, Renfro, and Payak 1970).

Disease severity can also be reduced by controlling the collateral weed host <u>Digitaria sanguinalis</u> where heavy growth of this weed occurs (Bains, Jhooty, and others 1978).

The pathogen can infect the embryo of the corn seed. Lowering the seed's moisture is a standard method of killing seedborne mycelia of downy mildews of corn. The seed should be ovendried for 24-48 hours at 35°C to reduce internal moisture to 8 percent and then stored in the desiccator for 40 days at this moisture level (B. P. Singh, personal communication 1984).

## Literature Cited

- Bains, S. S.; Jhooty, J. S.; and others. Role of <u>Digitaria</u> sanguinalis in outbreaks of brown stripe downy mildew of maize. Plant Dis. Rep. 62(2):143; 1978.
- Frederiksen, R. A.; Renfro, B. L. Global status of maize downy mildew. Ann. Rev. Phytopathol. 15:249-275; 1977.
- Payak, M. M.; Renfro, B. L. A new downy mildew disease of maize. Phytopathology 57(4):394-397; 1967.
- Payak, M. M.; Renfro, B. L.; Lal, S. Downy mildew diseases incited by Sclerophthora. Indian Phytopathol. 23(2):183-193; 1970.
- Shurtleff, M., Editor. Compendium of corn diseases. 2d ed. St. Paul, MN: American Phytopathological Society; 1980.
- Singh, J. P. Infectivity and survival of oospores of Sclerophthora rayssiae var. zeae. Indian J. Exp. Biol. 9(4):530-532; 1971a.
- Pathological and histopathological studies of brown stripe downy mildew of maize. Indian J. Exp. Biol. 9(4):493-495; 1971b.
- Singh, J. P.; Renfro, B. L. Studies on spore dispersal in Sclerophthora rayssiae var. zeae. Indian Phytopathol. 24(3): 457-461; 1971.
- Singh, J. P.; Renfro, B. L.; Payak, M. M. Studies on the epidemiology and control of brown stripe downy mildew of maize (Sclerophthora rayssiae var. zeae). Indian Phytopathol. 23(2):194-207; 1970.
- Singh, R. S.; Joshi, M. M.; Chaube, H. S. Further evidence of the seedborne nature of corn downy mildews and their possible control with chemicals. Plant Dis. Rep. 52(6): 446-449; 1968.

